KAREM SALEM

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Karem Salem : AKI CME Course :Mansoura/Ras EL Bar

CIN is one of leading causes of AKI in hospital setting

Table 2. Causes of Renal Insufficiency and Mortality by Cause

Cause	Episodes	Mortality
Decreased renal perfusion	147	20 (13.6)
Medications	61	9 (15)
Radiographic contrast media	43	6 (14)
Postoperative	35	1 (2.8)
Sepsis	25	19 (76)
Post–liver transplantation	14	4 (28.6)
Post–heart transplantation	8	3 (37.5)
Obstruction	7	2 (28.6)
Hepatorenal	7	5 (71.4)
Rhabdomyolysis	4	1 (25)
Artifactual	3	1 (33.3)
Glomerulonephritis	3	1 (33.3)
Nephrectomy	3	0 (0)
Atheroemboli	2	0 (0)
Hypercalcemia	2	1 (50)
Interstitial nephritis of unknown cause	2	0 (0)
Acetaminophen overdose	1	0 (0)
Unknown	13	1

NOTE. Values expressed as number or number (percent).

Table 5. Radiographic Contrast Studies Causing Renal Insufficiency

Cardiac catheterization and coronary angioplasty	21 (49)
CT scan	14 (33.3)
Peripheral angiogram	3 (7.1)
Endoscopic retrograde cholangiopancreatography	2
Pulmonary angiogram	1
Cholangiogram	1
Renal angiogram	1

NOTE. N = 43. Values expressed as number (percent).

Contrast-induced nephropathy is the third most common cause of in-hospital acute kidney injury and accounts for 10% of total cases.

published online February 4th, 2015

AGENDA

Definition
Pathophysiology
Types of contrast media
Risk Assessment
Risk Minimization
Fluids ??
Drugs ??
Dialysis ??

Definition of CIN

CI AKI

Table 1
The RIFLE and Acute Kidney Injury Network classifications of acute kidney injury

	GFR Criteria	Urine Output Criteria
*R*isk	Increased SCreat ×1.5 or GFR decrease >25%	UO <0.5 mL/kg/h × 6 h
*I*njury	Increased SCreat ×2 or GFR decrease >50%	UO <0.5 mL/kg/h × 12 h
*F*ailure	Increased SCreat ×3, GFR decrease >75% or SCreat >4 mg/dL (acute rise >0.5 mg/dL)	UO <0.3 mL/kg/h × 24 h or anuria × 12 h
*L*oss	Persistent AKI: complete loss of kidney function >4 weeks	
*E*SKD	End-stage kidney disease: complete loss of kidney function >3 months	
AKIN 1	Increased SCreat by 1.5–2× above baseline or by 0.3 mg/dL	UO <0.5 mL/kg/h × 6 h
AKIN 2	Increased Screat by 2–3× above baseline	UO <0.5 mL/kg/h × 12 h
AKIN 3	Increased SCreat by >3× above baseline or by ≥0.3 mg/dL in patients with baseline SCreat >4 mg/dL	UO <0.3 mL/kg/h × 24 h or anuria for 12 h

Abbreviations: AKI, acute renal failure; GFR, glomerular filtration rate; SCreat, serum creatinine; UO, urine output.

Definition of CIN
CI AKI

- **Increase in serum Cr compared to baseline**

>25% or 0.5 mg/dl increase of Cr level from baseline

- **Relationship between the rise of Cr and exposure to contrast agents**

Within 48-72 hrs of exposure

- **Exclusion of alternative explanations for renal impairment**

Cholesterol embolism, hypotension, UTI etc

Renal Atheroemboli

- Other signs of embolization (blue toes, livedo reticularis, abdominal pain)
- Transient eosinophilia and hypocomplementemia
- Renal failure which persists greater than 7 days

Pitfall in nephrology: contrast nephropathy has to be differentiated from renal damage due to atheroembolic disease

Journal of Nephrology, 05/18/2012 **Clinical Article**

Stratta P et al. – In a setting regarding millions of patients and millions of dollars/year, in order to clarify the true renal damage directly related to contrast medium (CM), the authors ask for prospective studies differentiating cohorts receiving intravenous and intra–arterial, transradial and transfemoral injections, and clinically relevant renal outcomes, thus avoiding the dangers that can come from the idolatry of a surrogate end point such an asymptomatic 25% transient increase of serum creatinine. To avoid that, patients may lose the possibility of a more useful radiological diagnosis, because of an exaggerated suspicion of risk.

CIN Timeline

- Symptoms 1-2 after exposure
- Creatinine peaks at 5-7 days
- Normalizes usually within 7-10 days

Acute Kidney Injury After Cardiac Surgery: Does the Time Interval From Contrast Administration to Surgery Matter

Journal of Cardiothoracic and Vascular Anesthesia, 04/25/2012

McIlroy DR et al. – In an appropriately selected population, cardiac surgery can be performed within 1day of cardiovascular catheterization and contrast administration without an increase in the incidence of postoperative AKI. Recommendations to delay cardiac surgery for a specified period after contrast administration to reduce the risk of postoperative acute kidney injury (AKI) are premature. Additional evidence is required before making recommendations on optimal surgical timing after contrast exposure.

National Kidney Foundation®

What is Nephrogenic Systemic Fibrosis (NSF)?

NSF is a rare but serious disease affecting skin and other organs that has been found in some patients with advanced CKD after exposure to gadolinium-containing contrast dyes that are used in magnetic resonance imaging (MRI). NSF appears to affect about 4 percent of patients with advanced CKD. People with **acute kidney injury (AKI)** are also at higher risk. NSF has not been reported in people with mild kidney damage or normal kidney function.

NSF can be painful, debilitating, or even fatal. Symptoms and signs of NSF can include burning and itching of the skin, red or dark patches on the skin, joint stiffness, or muscle weakness. The disease can develop within 24 hours up to around 3 months.

Contrast Agents

	Type	Iodine mg/ml	Osmolality mOsm/kg	cps	Viscosity
High Osmolar	• Ionic Monomer 2.75	325	1843		
	• Ionic Monomer	306	1530		5.0
Low Osmolar	• Ionic Dimer 7.5	320	580		
	• Non-ionic Monomer 4.7	300	616		
Iso Osmolar	• Non-ionic Dimer	300	320		8.1
	• Non-ionic Dimer 11.4	320	290		

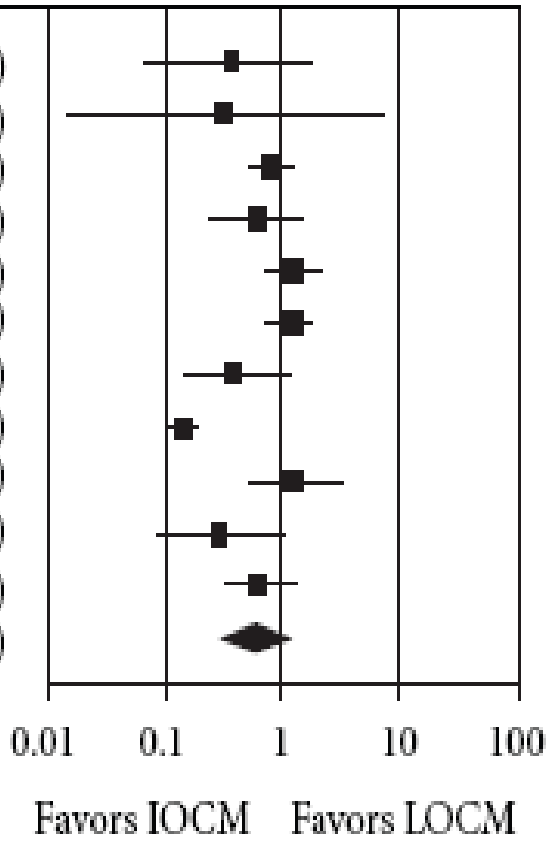
Contrast Agents

Contrast media	Molecular weight (Da)	Osmolality (mOsm/kg H_2O)	Iodine dose (mg/ml)	Iodine-atom-to-dissolved particle ratio	Viscosity (cPs at 37°)
High-osmolar ionic monomers					
Diatrizoate	636	1870	370	1.5	2.3
Ioxithalamate	643	2130	350	1.5	2.5
Low-osmolar non-ionic monomers					
Iopamidol	777	790	370	3.0	9.4
Iopentol	835	810	350	3.0	12
Iopromide	791	770	370	3.0	10
Iohexol	821	780	350	3.0	10.4
Ioversol	807	790	350	3.0	9
Iomeprol	778	620	400	3.0	12.6
Low-osmolar ionic dimers					
Ioxaglate	1270	600	320	3.0	7.5
<150 osmolar non-ionic dimers					
Iodixanol	1550	290	320	6	11.8
Iotrolan	1620	290	300	6	8.5

Contrast Media: Are There Differences in Nephrotoxicity among Contrast Media?

Richard Solomon

Study (year)	IOCM	LOCM	Rel. wt.	RR (95% CI)
Chalmers [30], 1999	54	48	6.88	0.356 (0.72–1.752)
Jakobsen [31], 1996	8	8	3.32	0.333 (0.016–7.140)
Wessely [16], 2009	162	162	11.00	0.801 (0.548–1.173)
Hardiek [15], 2008	54	48	9.49	0.625 (0.258–1.512)
Solomon [17] (CARE), 2007	210	204	10.59	1.265 (0.730-2.193)
Juergens [35], 2009	91	100	10.75	1.196 (0.733–1951)
Nie [37], 2008	106	102	9.04	0.398 (0.145–1.092)
Han [26] (abstract), 2010	828	828	11.23	0.147 (0.114–0.190)
Feldkamp [34], 2006	105	116	9.38	1.246 (0.500–3.109)
Hern ández [38], 2009	118	132	0.08	0.301 (0.085–1.063)
Mehran [40] (ICON), 2009	72	74	10.24	0.657 (0.337–1.280)
				0.577 (0.297–1.120)



Dye Volume

Diagnostic Cath : 100mL –

Interventional Cath: 250 – 300 mL –

Spiral CT Chest: 100 – 150 mL –

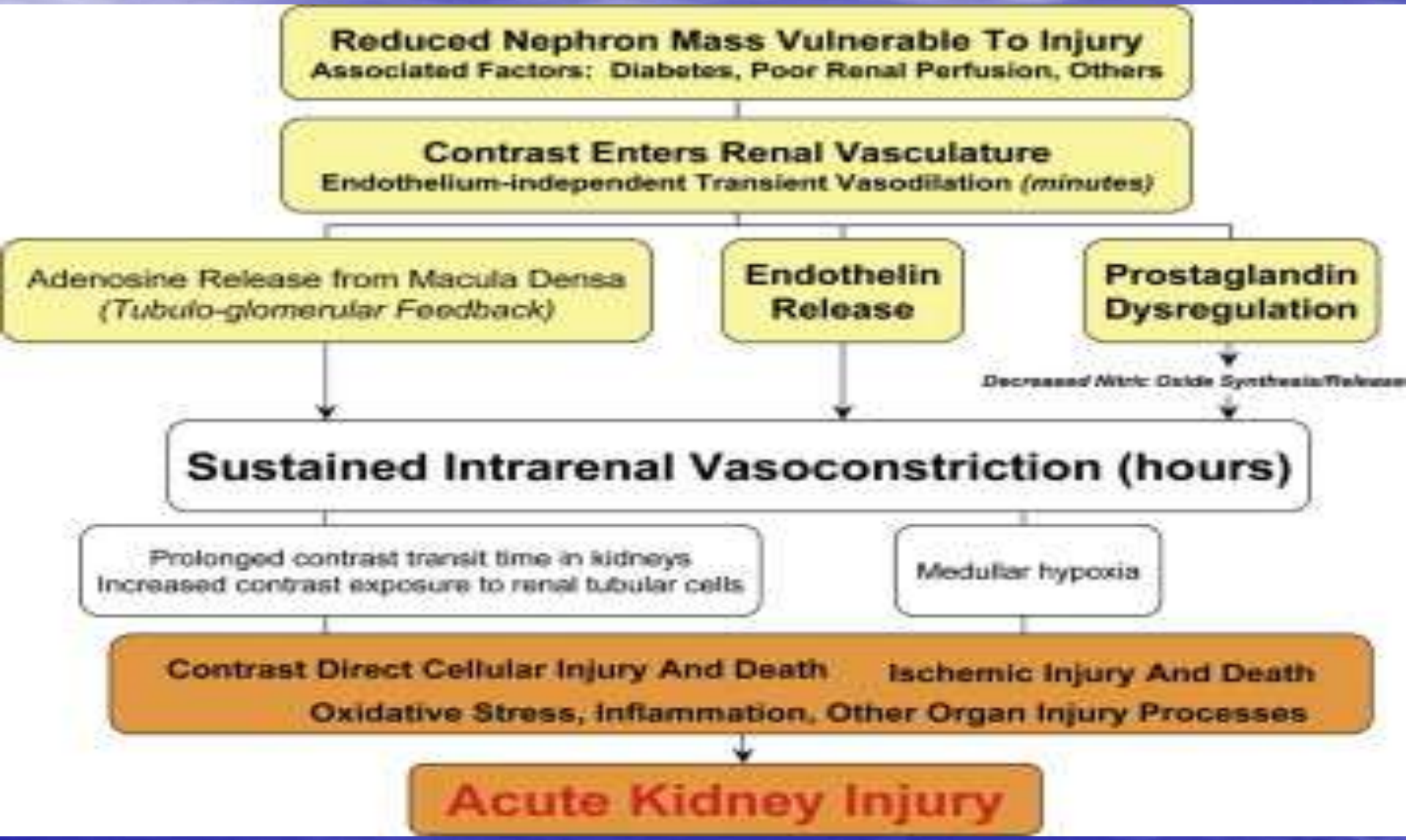
Cox et al. *Preventing Contrast Nephropathy: What Is the Best Strategy? A Review of the Literature . The Journal of Clinical Pharmacology, 2004; 44:327-337*

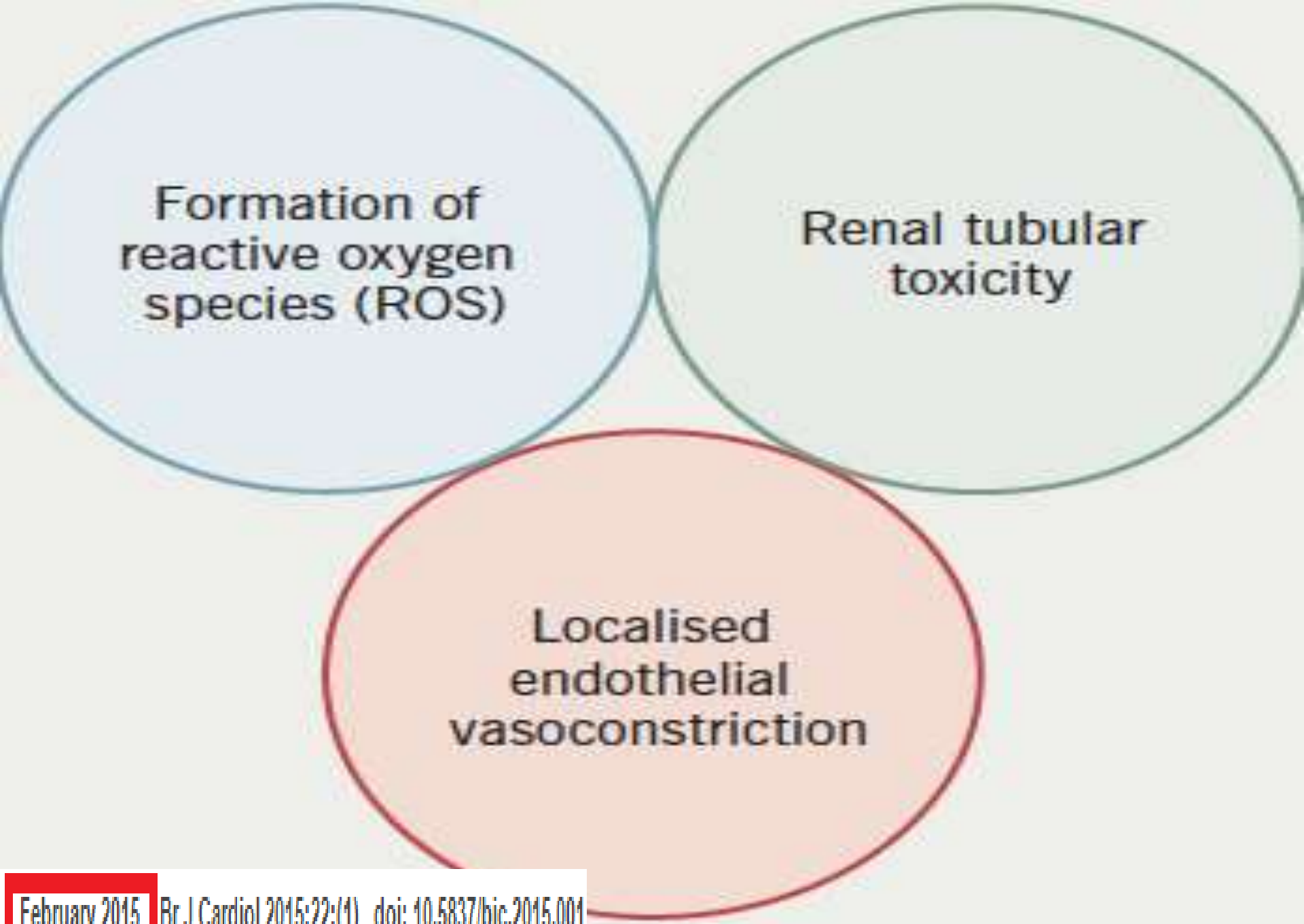
MAC : MRCD

$$\text{CM (ml)} = \frac{5 \times \text{BW(kg)}}{\text{sCr(mg/dl)}}$$

(Brown et al.,Circ Cardiovasc Interv. 2010)

Pathogenesis





February 2015 Br J Cardiol 2015;22:(1) doi: 10.5837/bjc.2015.001

Pathogenesis

- Ischemic acute tubular necrosis
  - Adenosine
  - Endothelin
  - Nitric oxide
  - Tubuloglomerular feedback (osmotic effects)
  - Direct endothelial damage
  - Smooth muscle injury
- Direct tubular toxicity
  - Tubular cell dysfunction and swelling due to hyperosmolarity
  - Reduced tubular cell ATP
  - Increased intracellular calcium concentration
- Oxidative stress and lipid peroxidation
  - Generation of reactive oxygen species
- Apoptosis of tubular cells
  - Tubular cell injury from osmotic stress
  - Hypoxia of tubular cells

Use of adenosine antagonist

Use of IVF / IOCM

Use of IOCM

Use of NAC

Use of IVF / IOCM

Risk Assessment

Table 1 | Risk factors for the development of CIN

Fixed (non-modifiable) risk factors	Modifiable risk factors
Older age	Volume of CM
Diabetes mellitus	Hypotension
Pre-existing renal failure	Anemia and blood loss
Advanced CHF	Dehydration
Low LVEF	Low serum albumin level (<35 g/l)
Acute myocardial infarction	ACE inhibitors
Cardiogenic shock	Diuretics
Renal transplant	Non-steroidal anti-inflammatory drugs
	Nephrotoxic antibiotics
	IABP

Abbreviations: ACE, angiotensin-converting enzyme; CHF, congestive heart failure; CIN, contrast-induced nephropathy; CM, contrast media; IABP, intra-aortic balloon pump; LVEF, left ventricular ejection fraction.

Risk Assessment

Risk Factors	Integer Score
Hypotension	5
IABP	5
CHF	5
Age >75 years	4
Anemia	3
Diabetes	3
Contrast media volume	1 for each 100 cc^3
Serum creatinine > 1.5mg/dl	4
OR	
eGFR <60ml/min/1.73 m^2	2 for 40 – 60 4 for 20 – 40 6 for < 20

eGFR < 60ml/min/1.73 m^2 = 186 x $(SCr)^{-1.154}$ x $(Age)^{-0.203}$ X (0.742 if female) x (1.210 if African American)

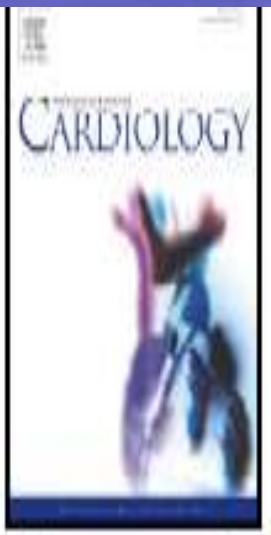
Calculate

Risk Score	Risk of CIN	Risk of Dialysis
≤ 5	7.5%	0.04%
6 to 10	14.0%	0.12%
11 to 16	26.1%	1.09%
≥ 16	57.3%	12.6%

Mehran et al. *JACC* 2004;44:1393-1399.

Risk Assessment

International Journal of Cardiology



A simple pre-procedural risk score for contrast-induced nephropathy among patients with chronic total occlusion undergoing percutaneous coronary intervention

International Journal of Cardiology 180 (2015) 69–71

Risk Assessment

In the past 3 months have you been told there may have been a change in your kidney function? Y/N

In the past 3 months have you been on any medications? Please list:

Have you used any over-the-counter pain relievers within the last 10 days? Y/N Please list:

In the past 3 months have you had any surgery? Y/N

Describe:

Do you feel dry or thirsty? Y/N

Circle one

*Have you ever been told you have kidney disease of any type? Please describe:	Y	N
*Have you had kidney surgery?	Y	N
*Do you have diabetes? Do you use insulin? Do you use metformin or glucophage?	Y Y Y	N N N
*Do you have hypertension, heart disease, or vascular disease?	Y	N
*Do you have gout?	Y	N
Do you have multiple myeloma?	Y	N
Have you ever had x-ray contrast media (dye) for CT, angiography, or IVP? Have you had contrast media within the last 3 days? Do you have any allergies to x-ray contrast media (dye)? Please describe:	Y Y Y	N N N
Have you received pretreatment with medication for this study?	Y	N
Do you have any allergies or asthma? Please describe:	Y	N

Risk Assessment

4. QUESTIONNAIRES
to be completed by clinicians referring patients for examinations using iodine- or gadolinium-based contrast media

1.	History of moderate or severe reaction to an iodine-based contrast medium	☐ Yes ☐ No
2.	History of allergy requiring treatment	☐ Yes ☐ No
3.	History of asthma	☐ Yes ☐ No
4.	Hyperthyroidism	☐ Yes ☐ No
5.	Heart failure	☐ Yes ☐ No
6.	Diabetes mellitus	☐ Yes ☐ No
7.	History of renal disease	☐ Yes ☐ No
8.	Previous renal surgery	☐ Yes ☐ No
9.	History of proteinuria	☐ Yes ☐ No
10.	Hypertension	☐ Yes ☐ No
11.	Gout	☐ Yes ☐ No
12.	Most recent measurement of serum creatinine	
	Value.....	
	Date	
13.	Is the patient currently taking any of the following drugs	
	Metformin	☐ Yes ☐ No
	Interleukin 2	☐ Yes ☐ No
	NSAIDs	☐ Yes ☐ No
	Aminoglycosides	☐ Yes ☐ No

World Kidney Day

14 March 2013

Kidneys for Life: Stop Acute Kidney Injury

www.worldkidneyday.org

Nonsteroidal anti-inflammatory agents[1,17,20,23]	Two deleterious effects: Interstitial inflammation[17] Decreased production of vasodilatory prostaglandins, thereby potentiating the effect of adenosine, increasing vasoconstriction[17,20]
Calcineurin inhibitors[1]	Vasoconstriction of afferent arterioles, resulting in irreversible damage; glomerular sclerosis[20]
Loop diuretics[17,23]	Risk of volume depletion before procedure[17] After procedure, forced euvolemic diuresis associated with increase in risk for contrast-induced nephropathy[2,16,18]
Aminoglycosides[1,9,17,20,23]	Intracellular accumulation of medication in proximal tubule cells thought to interfere with cellular function, eventually leading to cell death and decreased glomerular filtration rate[9,20]
Amphotericin B[5,17,20,23]	Binds to tubular epithelial cells, alters cell permeability, and causes vasoconstriction of intrarenal arteries and arterioles[9]
Vancomycin[5,17]	Mechanism of nephrotoxic effect is unknown[5,18]
Chemotherapeutic agents[20,23]	Cumulative nephrotoxic effects related to tubular epithelial cell necrosis[23]
Metformin[1]	Not a nephrotoxic medication; however, has been associated with spontaneous lactic acidosis resulting in acute kidney injury, systemic complications, and death[1,18] May be reinstituted after renal function is normal[18,19]

Risk Minimization

Table 1 Summary of strategies/agents evaluated to prevent CIN

Strategies/agents shown to have DEFINITIVE value
- Parenteral hydration
- Low volume of contrast medium
- Low-osmolar and iso-osmolar contrast media
- Employ noniodinated contrast studies

Strategies/agents of POSSIBLE value
- Epogen
- Sodium bicarbonate
- NAC
- Theophylline
- Hemofiltration
- Iloprost
- Mesna
- Acetazolamide
- Protacyclins
- Nebivolol

Strategies/agents of DOUBTFUL value
- Atrial natriuretic peptide
- Fenoldopam
- Dopamine
- Mannitol
- Furosemide
- Nifedipine
- Endothelial nonspecific receptor antagonists
- Hemodialysis
- Statins
- Ascorbic acid

Canadian Association of Radiologists: Consensus Guidelines for the Prevention of Contrast-Induced Nephropathy

ESUR Guidelines on **Contrast Media**

ACR Manual on Contrast Media

ACR
American College of Radiology
Quality is Our Image

Version 9
2013

BJC
The British Journal of Cardiology

February 2015 Br J Cardiol 2015;22:(1) doi: 10.5837/bjc.2015.001

Contrast-induced nephropathy in PCI: an evidence-based approach to prevention

4.1: Define and stage AKI after administration of intravascular contrast media as per Recommendations 2.1.1-2.1.2. (*Not Graded*)

4.1.1: In individuals who develop changes in kidney function after administration of intravascular contrast media, evaluate for CI-AKI as well as for other possible causes of AKI. (*Not Graded*)

4.2.1: Assess the risk for CI-AKI and, in particular, screen for pre-existing impairment of kidney function in all patients who are considered for a procedure that requires intravascular (i.v. or i.a.) administration of iodinated contrast medium. (*Not Graded*)

4.2.2: Consider alternative imaging methods in patients at increased risk for CI-AKI. (*Not Graded*)

4.3.1: Use the lowest possible dose of contrast medium in patients at risk for CI-AKI. (*Not Graded*)

4.3.2: We recommend using either iso-osmolar or low-osmolar iodinated contrast media, rather than high-osmolar iodinated contrast media in patients at increased risk of CI-AKI. (*1B*)

4.4.1: We recommend i.v. volume expansion with either isotonic sodium chloride or sodium bicarbonate solutions, rather than no i.v. volume expansion, in patients at increased risk for CI-AKI. (*1A*)

4.4.2: We recommend not using oral fluids alone in patients at increased risk of CI-AKI. (*1C*)

4.4.3: We suggest using oral NAC, together with i.v. isotonic crystalloids, in patients at increased risk of CI-AKI. (*2D*)

4.4.4: We suggest not using theophylline to prevent CI-AKI. (*2C*)

4.4.5: We recommend not using fenoldopam to prevent CI-AKI. (*1B*)

4.5.1: We suggest not using prophylactic intermittent hemodialysis (IHD) or hemofiltration (HF) for contrast-media removal in patients at increased risk for CI-AKI. (*2C*)

Hydration

Mechanism

- Dilute CM in the tubules
- Decrease contact time
- Increase production of prostacycline in renal medulla

BICARB : Increase pH of urine and renal medulla

- Suppress production of free radicals

- Volume expansion
  - Suppress renine-angiotensine system
  - Suppress ADH

The incidence of contrast nephropathy was significantly lower in patients with urine pH >6.0 immediately before coronary angiography compared with patients with urine pH >6.0 ($P < 0.0001$) (Table 3).

Efficacy

4.4.1: We recommend i.v. volume expansion with either isotonic sodium chloride or sodium bicarbonate solutions, rather than no i.v. volume expansion, in patients at increased risk for CI-AKI. (*1A*)

4.4.2: We recommend not using oral fluids alone in patients at increased risk of CI-AKI. (*1C*)

N Am J Med Sci. 2014 Dec; 6(12): 618–624.
doi: 10.4103/1947-2714.147977

Oral Hydration for Prevention of Contrast-Induced Acute Kidney Injury in Elective Radiological Procedures: A Systematic Review and Meta-Analysis of Randomized Controlled Trials

Sodium chloride vs. sodium bicarbonate for the prevention of contrast medium-induced nephropathy: a randomized controlled trial

European Heart Journal, 01/31/2012

Klima T et al. – Volume supplementation with 24 h sodium chloride 0.9% is superior to sodium bicarbonate for the prevention of contrast–induced nephropathy (CIN). A short–term regimen with sodium bicarbonate is non–inferior to a 7 h regimen.

Int Urol Nephrol. 2015 Feb;47(2):321-6. doi: 10.1007/s11255-014-0820-0. Epub 2014 Aug 28.

Sodium bicarbonate infusion for prevention of acute kidney injury: No evidence for superior benefit, but risk for harm?

Schiffl H[1].

[1]Hydration with Saline Guidelines

IVF = 1 mL/kg/hr (**MAX** 100 ml/hr) **12** hours pre & **12** hours post contrast* (24 hour total infusion duration)
(*NS preferred IVF but MD can modify based on clinical status of patient)

CHF or left ventricular ejection fraction (LVEF) < 40%?
0.5 ml/kg/hr (max 50 ml/hr) 12 hrs pre & post contrast (24 hour total infusion duration)

Emergent procedure? (suggested regimen):
Fluid bolus of 500-1000 ml prior to procedure. Hydration during procedure and/or 12 hrs after if possible (dependent on clinical status)

[2]Bicarbonate Dosing Guidelines

IVF = 150 meq of sodium bicarbonate in **1** liter of D5W

3 ml/kg bolus (**MAX** 300 ml) **1** hour prior to procedure AND **1** mL/kg/hour (**MAX 100** ml/hr) during and for 6 hours post-procedure

Glycemic control issues (including patients with diabetes)?
Consider mixing sodium bicarbonate in 1 liter of sterile water instead of D5W

Furosemide with Saline Hydration for Prevention of Contrast-Induced Nephropathy in Patients undergoing Coronary Angiography: A Meta-Analysis of Randomized Controlled Trials

MEDICAL SCIENCE MONITOR

Med Sci Monit 2015; 21: 292-297

Conclusions

Furosemide treatment appears to have no additional influence beyond saline hydration on the incidence of CIN and subsequent dialysis after a coronary interventional procedure.

N-Acetyl Cysteine

Mechanism

Oxygen Free Radical Scavenger & Vasodilator

- Advantages :
  - ❖Low cost
  - ❖Ease of administration
  - ❖Limited side-effects

Efficacy

- ○Optimal dosage remains uncertain (600 mg b.d or 1200 mg b.d for 48 hr)
- ○Exact mechanism to prevent CIN is unknown
- ○Induces creatininuria leading to a reduction in serum creatinine independent of a change in GFR

4.4.3: We suggest using oral NAC, together with i.v. isotonic crystalloids, in patients at increased risk of CI-AKI. (2D)

N-Acetyl Cysteine

[3]Acetylcysteine Dosing Guidelines

Tolerating PO intake?
600-1200 mg capsules PO Q12h X 4 doses
2 doses pre-contrast and 2 doses post-contrast is optimal

Feeding tube or NG-access?
Acetylcysteine 600-1200 mg (3 mL of 20% soln.) liquid PT/NG Q12h x 4 doses total

Emergent Procedure?
1 dose before and 3 doses post cath or procedure is acceptable (Q12h x 4 doses total)

Theophylline

Mechanism

Adenosine Antagonist

Efficacy

4.4.4: We suggest not using theophylline to prevent CI-AKI. (2C)

Statins

Mechanism

Beneficial effects on endothelial function.
Maintain NO production and ↓oxidative stress.

Efficacy

- Not enough evidence

J Clin Pharmacol. 2015 Feb;55(2):123-31. doi: 10.1002/jcph.411. Epub 2015 Jan 5.

Meta-analysis of short-term high versus low doses of atorvastatin preventing contrast-induced acute kidney injury in patients undergoing coronary angiography/percutaneous coronary intervention.

Ascorbic Acid

Mechanism

Prevention of oxidative stress

Efficacy

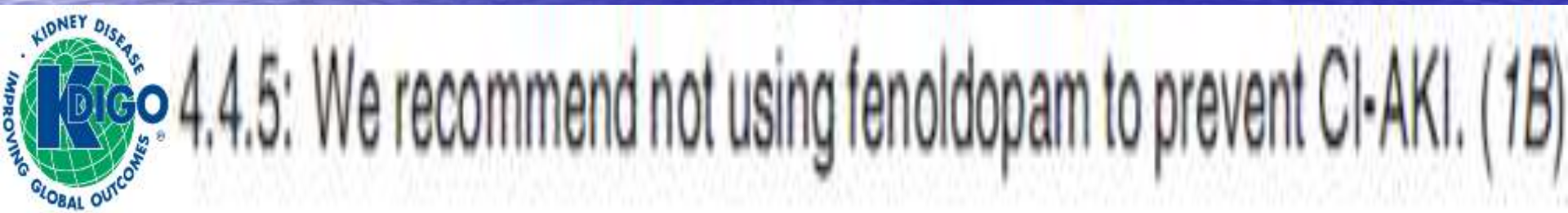
- 3g before and 2g x2 post procedure
- Safe & well tolerated
- Not enough evidence

Dopamine / Fenoldopam

Mechanism

Renal Vasodilator

Efficacy



Hemodialysis For Prevention Of CIN

- **Few Studies with Results not easy to compare**
  - **Differences in patients selection.**
  - **Differences in extracorporeal treatment modalities.**
  - **Differences in dose of contrast medium infused.**
  - **Timing of extracorporeal treatment with respect to contrast administration**

Efficacy

4.5.1: We suggest not using prophylactic intermittent hemodialysis (IHD) or hemofiltration (HF) for contrast-media removal in patients at increased risk for CI-AKI. (*2C*)

Contrast Media In Dialysis Patients

Dilution Hyponatraemia

Increase in extra-cellular osmolality by contrast medium (causes watermigration from the intra-cellular to the extra-cellular compartment).

Hyperkalaemia

may be due to potassium migration from the intracellular to the extracellular compartment.

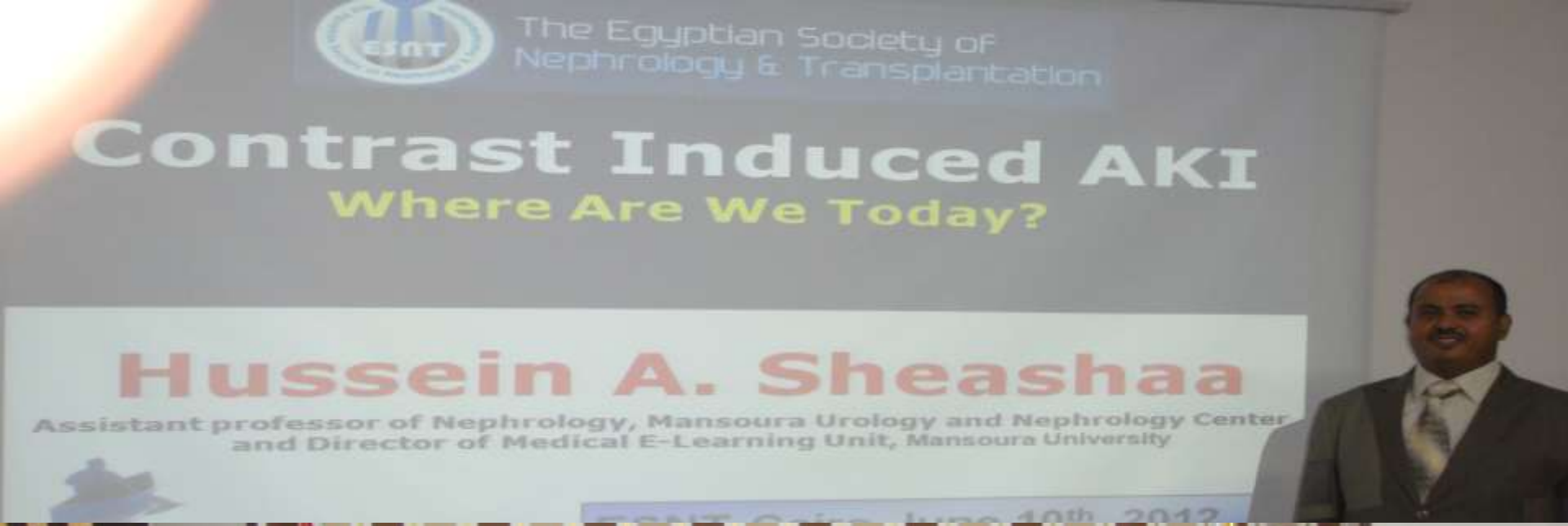
Acute pulmonary oedema.

particularly in those with volume overload or chronic heart failure and poor cardiovascular stability.

The role of dialysis in contrast-induced nephropathy: doubts and certainties

Carlo Guastoni[a], Stefano De Servi[b] and Marco D'Amico[c]

- The administration of contrast media should be better done the subsequent day following dialysis treatment, when the patient is at his/her dry body weight.
- The dose of contrast medium administered should be as low as possible (possibly <150 ml).
- Avoiding contemporary diagnostic and therapeutic procedures.


The Egyptian Society of
Nephrology & Transplantation
Contrast Induced AKI
Where Are We Today?
Hussein A. Sheashaa
Assistant professor of Nephrology, Mansoura Urology and Nephrology Center
and Director of Medical E-Learning Unit, Mansoura University

CIN in 1 Slide

1-risk scoring the pt is at ...%risk for develop CIN
& .....% need for dialysis

2- if high risk u should consider another modality for imaging.

3-preventive measures

a-DC any diuretics. ACEI,NSAIDS 3days before the procedure

b-no fasting night before procedure

c-saline 0.9% 1ml/kg 12h pre & 12 hrs post

d- emergency 3ml isotonic bicarb 1h pre & 1ml/kg 6h post

e-oral NAC 600 mg 12h pre &36h post

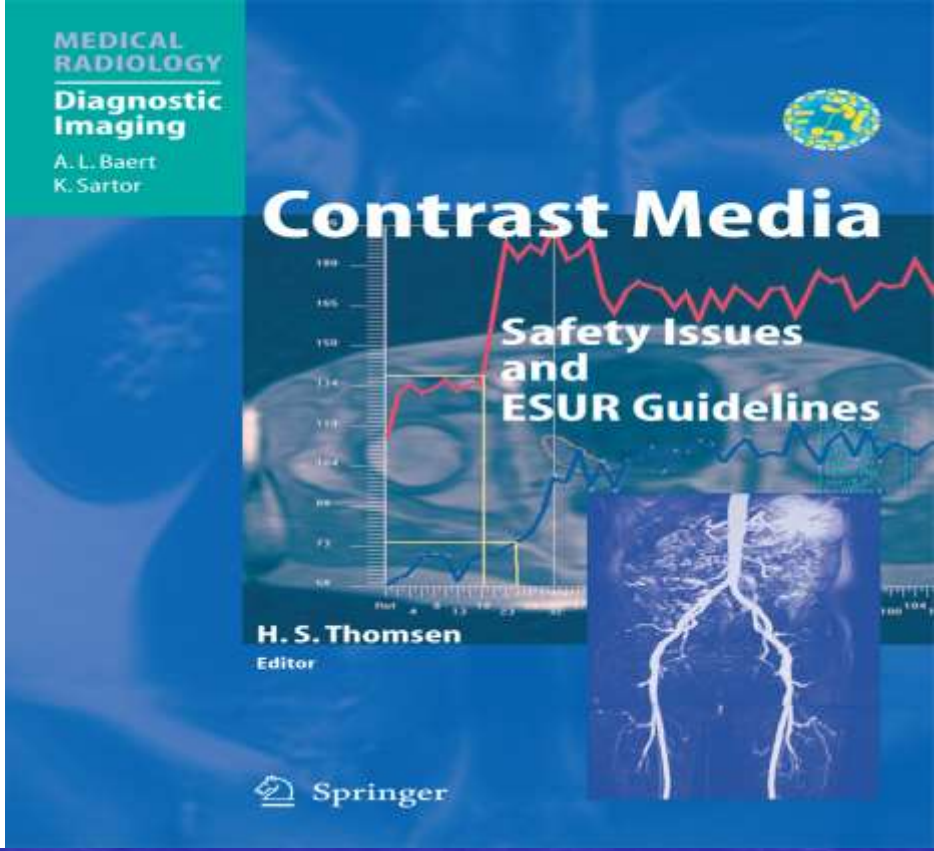
your MRCD -BW×5/S.CREAT- .... ML

use non ionic low or isoosmolar contrast

3- follow up urine out put after if ≥150 ml/hr u r doing well.....

4-monitor renal functions daily for 1 week


THANK
YOU SO
MUCH!



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